# *Modern Concepts of Cardiovascular Disease*

*Published monthly by the* AMERICAN HEART ASSOCIATION
50 WEST 50TH STREET, NEW YORK, N. Y.

DR. SAMUEL A. LEVINE, Boston, *Editor*
DR. MARSHALL N. FULTON, Boston, *Associate Editor*

**Vol. IV** **October, 1935** **No. 10**

## AURICULOVENTRICULAR HEART BLOCK

AURICULOVENTRICULAR heart block is a symptom and not a disease. It is classified as an arhythmia, since it is a disturbance of the orderly mechanism of the heart beat. The difficulty lies in the inability of the conduction system to carry the impulses from the auricles to the ventricles normally. Physiologically, A-V heart block occurs in auricular fibrillation, auricular flutter and excessively rapid auricular paroxysmal tachycardia, since the auricular impulses are "bombarding" the A-V node more rapidly than the node and bundle can normally conduct them. In auricular fibrillation only the stronger impulses are carried through. In rapid auricular flutter or tachycardia (over 200 beats per minute) the ventricles usually respond to every other auricular beat.

In pathologic heart block the difficulty may range from a simple sluggishness of conductivity to complete inability of the junctional tissues to conduct. Simple retardation (first degree block) can be recognized only by graphic methods. Higher degrees of block are manifested by an occasional dropped beat or by regularly recurring failure of contraction of the ventricles (second degree block) or by total dissociation of auricular and ventricular contractions; complete heart block. Partial heart block is much more common than complete block. In the higher grades of partial heart block, dropping of the beats recurs regularly, every fourth auricular impulse failing to travel down the junctional tissues to the ventricles (4 to 3 block), or every third (3 to 2 block), or every second (2 to 1 block), or every second and third (3 to 1 block). Higher grades of partial heart block are rare, idioventricular rhythm usually being established when the block becomes greater than 2 to 1.

When the ventricles beat independently of the auricles they do so at rates between 25 and 30 per minute, and in infants and young children between 50 and 70 per minute. The auricles contract at their ordinary rate, although they respond normally to accelerating or retarding influences and are occasionally subject to arhythmias, such as auricular premature beats and auricular fibrillation. The ventricles show relatively little variation in rate of contraction. Occasionally there is an alternation between complete heart block and high-grade partial block. In advanced cases of complete block periods of ventricular standstill of variable duration may occur. Such episodes have been known to last as long as five minutes (Levine and Matton) with temporary recovery, but much shorter ones are the rule. In rare instances in complete block, the ventricles beat more rapidly than the auricles.

Auriculoventricular heart block may be temporary and functional. The functional cases are usually incomplete and are always transient or recurrent, never permanent. The commonest causes of functional block are digitalis or quinidine poisoning, asphyxia, excessive vagal stimulation, uremia and infectious diseases, mainly rheumatic fever and diphtheria.

Acceptable researches on the structural basis of organic heart block have been relatively few. The entire conduction system must be studied by serial sections. This includes the A-V node and bundle and the bundle branches as far at least as the bases of the papillary muscles. Considerable experience is necessary in this field of research. Every case of established or long-standing heart block, at least of high grade, is due to organic changes in the conduction system above the bases of the papillary muscles. All of the cases of established heart block reported to have been free of organic lesions in the conduction system have presented some serious flaw in the investigation. The most common shortcoming has been a failure to study the bundle branches.

THE commonest cause of organic heart block is fibrosis of the bundle of His or of both bundle branches. This is practically always on a vascular basis. There is diminution in blood supply due to sclerosis of either the larger coronary arteries or the smaller intramyocardial branches. The A-V node and bundle are supplied with blood almost entirely by one special artery, the *ramus septi fibrosi*, or first posterior perforating artery, a branch from the point of origin of the posterior descending branch of the right coronary artery. In the main, the right bundle branch and the anterior division of the left bundle branch are supplied by perforating septal branches of the anterior descending artery, a branch of the left coronary artery. The posterior division of the left bundle branch is supplied by similar septal branches of the posterior descending artery. A common cause of heart block seldom mentioned is fibrosis of both bundle branches, frequently with little change in the surrounding myocardium. The conducting tissues are apparently more susceptible to diminution of blood supply than the myocardium.

The next most common organic cause of heart block is localized calcification of the interventricular septum, usually at the junction of its membranous and muscular portions where the bundle of His is located. This deposit of calcium is usually due to strain at that point and not primarily to vascular disease. The commonest causes of organic heart block occur, therefore, in older people, more often in men.

Other organic causes are relatively rare. Chief among these are gumma of the interventricular septum and congenital defects of the septum that produce an

interruption in the course of the bundle of His. Tumors of the septum, of the A-V node or bundle, endocarditis, aneurysm of the septum, fatty degeneration of the A-V node, idiopathic myocarditis and infarction of the septum are rare organic causes. In general, it may be stated that permanent heart block in children is usually due to a congenital defect of the septum, in the prime of life to a gumma in the septum, in middle age to coronary artery sclerosis, and in old age to a calcium deposit in the septum.

One reason for the relative rarity of heart block is the great damage the conduction system may suffer without complete heart block resulting. Apparently a few strands are sufficient to conduct impulses more or less normally.

The site of the new ventricular pacemaker in complete heart block is assumed to be the highest point in the conduction system below the obstruction. When the lesion is in the A-V node or bundle, therefore, the ventricular complexes of the electrocardiogram are similar in form to normal complexes (supraventricular type). When the lesions are confined to the bundle branches the pacemaker is probably in the course of one or the other branch, and the ventricular complexes frequently resemble those of "bundle branch block." Often in such cases the form of the complex varies from time to time, shifting from forms suggesting first a block in one branch and then in the other. This transition is due to a shift in the site of the pacemaker from one ventricle to the other. When the ventricular rate is rapid the pacemaker is probably an ectopic focus in one of the ventricles, and the organic block is probably low in the course of both bundle branches. In cases of shift in the degree of block or of alternation between sinus rhythm and some degree of block, varying vagal influence or a transient toxic factor probably enter the picture. Periods of asystole or ventricular standstill may be so explained, or an additional circulatory factor affecting the pacemaker may be assumed to exist.

All forms of heart block are demonstrable by electrocardiograms. First degree A-V block is shown by simple prolongation of the P-R interval beyond 0.2 second. When there is progressive lengthening of the P-R interval a dropped beat results. After the dropped beat the P-R interval is shorter but again becomes progressively prolonged with subsequent beats. When there is regular dropping of the beats at short intervals this variation in the P-R interval is slight. With complete A-V dissociation there is no relationship between the auricular and ventricular complexes, and the auricles and ventricles contract independently at their own rate, usually with regular rhythm.

SYMPTOMS caused directly by heart block are few and rarely observed. Palpitation is occasionally complained of. It is only when there is a sudden change in the degree of block or when transient periods of asystole occur that serious symptoms appear. Then there is evidence of cerebral anemia ranging from slight giddiness to unconsciousness and even convulsions. Such attacks are designated as the Adams-Stokes syndrome.

With lesser grades of block, exercise and excitement either speed up the ventricular rate or decrease it. Slowing of the rate is due to an increase in the degree of block because of the accelerated auricular rate. With complete block the ventricular rate is usually constant or changes only slightly.

On physical examination the delay between auricular and ventricular beats may be noted occasionally in the jugular venous pulse. On auscultation an extra sound may be heard due to separation of auricular and ventricular contractions. A systolic apical murmur is not uncommon. When the ventricular rate is very slow the systolic blood pressure is usually moderately elevated. This is the compensatory effect of extra filling and complete emptying of the ventricles. Even in complete block the blood flow at rest may be normal because of the increased output of blood per beat.

Fluoroscopic examination allows visual demonstration of independent auricular and ventricular activity. Usually there is some cardiac enlargement.

Heart block may exist for years with very little impairment of activity of the patient. The prognosis is somewhat more grave in the elderly than in the young, particularly when the block is permanently established. Lesser grades of block may be of more serious import than chronic complete block. Complete block may exist for a long time before Adams-Stokes attacks occur. These may increase in frequency and severity, or they may disappear after a time. Occasionally a patient dies as the result of ventricular standstill. Occurring in the course of an acute infection, heart block adds gravity to the prognosis. Congestive heart failure and angina pectoris may be complications in cases of heart block. In general, the prognosis is dependent not so much on the presence of the block as on the integrity of the myocardium and the occurrence of Adams-Stokes attacks.

In the treatment of the patient with heart block the underlying cardiac disease should receive first attention. Salicylate therapy may be of value in acute rheumatic heart block, antiluetic therapy in suspected cases of gumma of the septum, omission of toxic agents in cases of drug poisoning, the usual treatment when congestive failure exists, and the measures ordinarily employed when coronary artery sclerosis appears to be present. When congestive failure occurs digitalis may be used provided the heart block has not been originally produced by the drug.

In preventing or treating attacks of the Adams-Stokes syndrome, by far the most efficacious drug is epinephrine (adrenalin) hydrochloride in doses of 0.5 to 1 cc. of the 1:1000 solution injected subcutaneously or intravenously. This may be given every few hours when the attacks recur rapidly. The drug acts either by stimulating the new pacemaker or by decreasing the grade of block. Instead of epinephrine, ephedrine hydrochloride in doses of ⅜ to ½ grain (20 to 30 mg.) taken orally or given by injection hypodermically may be employed as preventive therapy. If epinephrine or ephedrine prove ineffectual, thyroid extract 1½ grains, (0.1 gram) three times a day or barium chloride ½ to 1 grain, (0.03 to 0.06 gram) three or four times a day may be tried. These drugs occasionally prevent attacks by exciting the ventricles to more rapid action. Thyroid extract, however, usually increases the rate of the auricles without affecting the ventricles. Of least value is atropine sulphate 1/120 to 1/60 grain, (0.5 to 1 mg.) subcutaneously or orally every few hours. It occasionally lessens the degree of block when excessive vagal tone plays a role. Intracardiac injection of epinephrine may be life-saving when death impends from ventricular standstill.

WALLACE M. YATER, M.D., Washington, D. C.